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The University of Toronto Medical Journal

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EDITORIAL.

This issue introduces the fifth volume of the University of Toronto Undergraduate Medical production. We trust that it will continue to meet with the well-merited success which it has enjoyed in the past.

The Journal's prime object is to offer an opportunity to the Medical Student to write papers. Confidence in preparing material for publication is a vast aid to the physician. We shall endeavour this year to emphasize this phase and urge the co-operation of the student body in offering contributions to their journal. It is particularly hoped that the Journal in part at least may be interesting to the first, second and third years as well as valuable to file for the clinical years.

Apart from being a means of student expression, the Journal fills a large place in giving us the ideas of our staff in concrete form. It is well recognized that Toronto possesses a Medical Faculty second to none. The articles of its professors are invaluable to student and practioner alike; they present in synopsis form the recent and worth-whlie advances in Medical Science. It is largely for this reason that the Journal is regularly taken by a rapidly increasing number of practising physicians in all parts of the country; the journal will welcome contributions from these men.

We thank the staff and students for their interest and assistance in the past and trust that we will all combine to make this volume the most useful one yet produced.

The death of the late Miss Mary Cairo, in July, 1927, is recalled with regret by medical undergraduates.

Miss Cairo had just completed the fifth year of her course. Consequently she was not far from the goal which was to enable her to fulfil a unique position among the Italian citizens of Toronto.

Her contributions, to student life, have made the realization of her death doubly poignant. How many times have her classmates listened, with keen enjoyment, to her sympathetic interpretations on the piano! For her music, for her courage and ambition, for herself, she was loved. We think the formula used by the gods for Mary's personality, must have called for a dash of golden Italian sunlight. How else can one account for a certain quality, that was hers, the memory of which will long be cherished by those who knew her?

To the members of her family, and her friends, who are many, we should like to extend our sincere sympathy.

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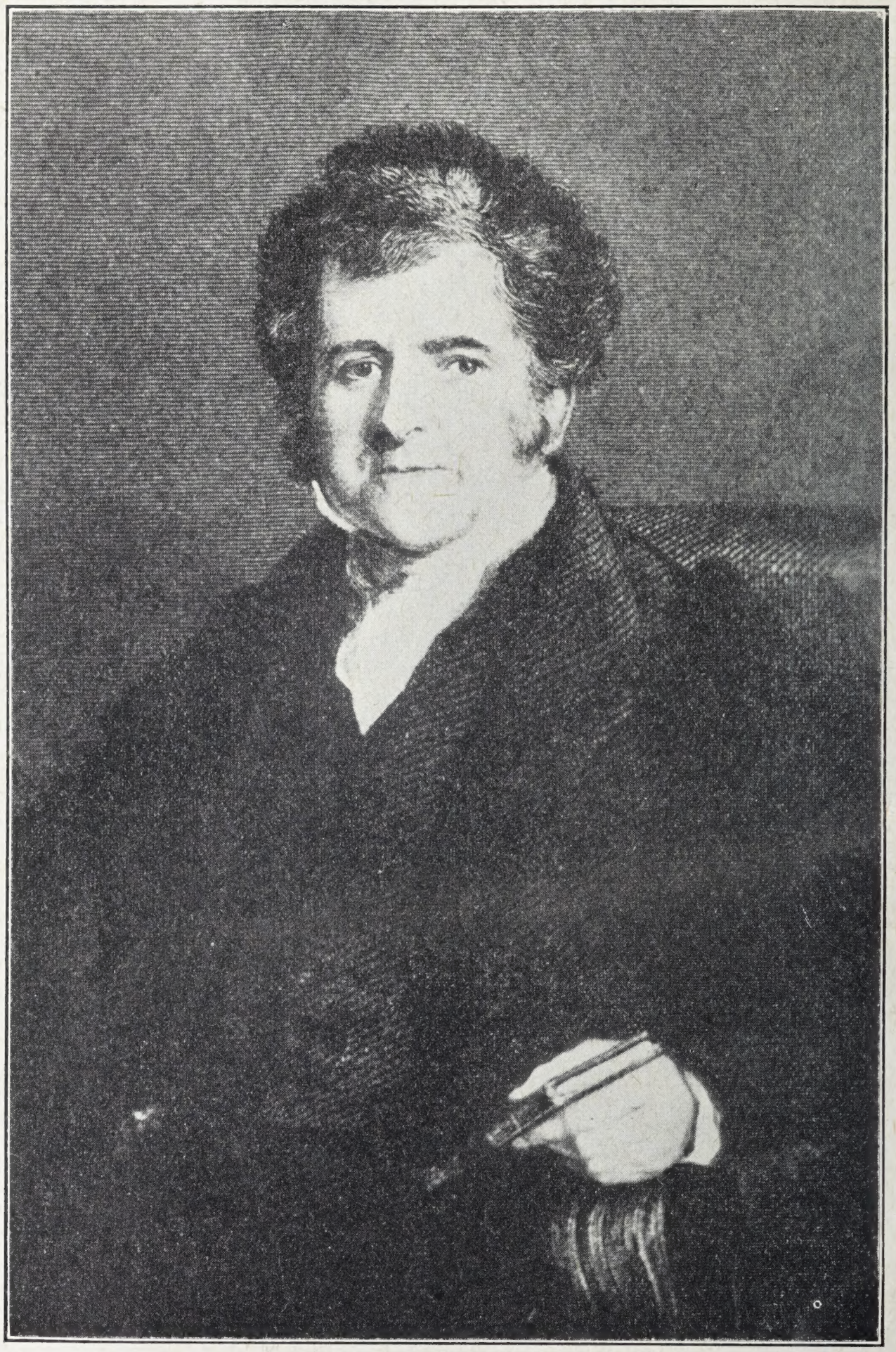
It is with regret that we announce the retirement of Berton Yuill from the position of Business Manager of the Medical Journal. For the past two years Mr. Yuill's enthusiastic and untiring services have formed a large factor in placing the Journal in its present sound position. We congratulate Mr. Yuill in obtaining the opportunity of pursuing his studies in London, England, and wish him every success.

We are fortunate in having as his able successor, Mr. G. Grant. Under his ministrations we are sure that the Journal will continue to thrive.



RICHARD BRIGHT, M.D.

Physician, Guy's Hospital, London, 1820-1843.

The Story of Richard Bright.

(Ben I. Johnstone, M.B. B.Sc.)

Just one hundred years ago, Richard Bright first published his observations on kidney disease, which were to revolutionize the existing ideas regarding dropsy and albuminous urine, and to make their author famous. His work was by no means confined to the kidney, nor were his interests related only to medicine. It was, however, as a direct result of these investigations that his name has been perpetuated in "Bright's disease," and is familiar to every student of medicine even to-day.

The latter part of the eighteenth, and the early part of the nineteenth centuries, form the golden age in clinical medicine. This is amply proven by the remarkable activity in clinical and pathological investigations in various countries during that time.

In France, we are familiar, particularly, with the work of Louis and Laennec. Rokitansky and Skoda were the most prominent workers in the New Vienna School, while in Dublin, at this time resided that remarkable group of men, who made that school so famous, namely, John Cheyne, Abraham Colles, Robert Adams, William Stokes, Dominic John Corrigan and Robert James Graves.

In London, of special importance was the clinical and pathological work, which was being done by the long line of brilliant workers at Guy's Hospital. Of these Richard Bright was not the least outstanding. In fact, there has been no English physician, perhaps, with the single exception of Laennec, it may be said, none of any country since the time of Harvey, who has effected, not only so great an advance in the knowledge of particular diseases, but also so great a revolution in our habits of thought and methods of investigating morbid phenomena, as did Richard Bright.

He was born at Queen's Square, Bristol, on Sept. 28, 1789, the third son of Richard Bright, Senior, a prosperous merchant and banker of that city. The father belonged to the Brights of Brockbury, Herefordshire, who trace their descent to a most learned, pious and eloquent man, Henry Bright, D.D., of Brockbury, who was Master of King's School at Worcester in Queen Elizabeth's time.

Bright's father later lived at Ham Green, in the county of Somerset, while his eldest brother represented the city of Bristol in three successive parliaments.

After having passed through the usual course of elementary education, first, at a private school in his native city, and afterwards under a tutor, in Exeter, Richard Bright matriculated in the autumn of 1808, at the University of Edinburgh, at the age of nineteen. During his first year, he devoted his attention exclusively to the general lectures at the University, amongst which were those of moral philosophy and political economy by Dugald Stewart, natural philosophy by Playfair, and mathematics by Leslie; in the higher class of which, he that year successfully competed for the prize.

In the session of 1810, he pursued the studies more immediately connected with his chosen profession, and besides attending the course in chemistry by Hope, and lectures in anatomy by Munro, and medicine by Duncan, he availed himself of the excellent anatomical course delivered out of college by Dr. John Gordon.

In the summer of 1810, Sir George Mackenzie, a noted mineralogist, planned a trip to Iceland. He chose a young doctor by the name of Holland, and Richard Bright to accompany him. The account of this interesting tour was published by Mackenzie, the following spring, to which Dr. Holland contribut-

ed an important chapter on the literature, history, laws, religion and diseases of the Icelanders. Bright contributed a general sketch of the zoology and botany of the island.

Though this trip must have been a serious interruption to his professional studies, it, doubtless, had its use in training his great powers of observation.

Mackenzie describes Holland and Bright as "young men of very superior talents and requirements and in a high degree pleasing in manner." Holland constantly refers to Bright as an ideally cheerful companion, who put up with all the mishaps and hardships of the trip with good humour and equanimity.

Holland had known Bright at Bristol in 1803. The former spent two winters at Guy's and St. Thomas' hospitals in London, took his M.D. from Edinburgh, and in 1811 had spent a year and a half travelling on the continent, especially in Austria and Hungary. There can be little doubt that Bright had Holland in his mind when he carried out his programme of studies for the four years following his trip to Iceland.

On returning, he pursued his studies in London, living for two years in the house of one of the resident officers of Guy's Hospital. Here he attended lectures on the practice of medicine by Dr. Wm. Babington and James Currie, and studied anatomy and surgery under Sir Astley Cooper, the two Clines and Travers. It is supposed that from Astley Cooper Bright imbibed a sense of the value of morbid anatomy in the study of diseases. Even at that time, he executed a drawing, since preserved, of the appearance of the kidney in the malady, by the investigation of which he, afterwards, made himself famous.

Not satisfied with a mere knowledge of those branches of science which bore on his profession directly, his active mind delighted in excursions into those collateral sciences, geology, zoology and botany, which may be considred as the ornamental part of a medical education, without which a man may be a good practitioner but cannot be an accomplished physician.

In these studies Bright made considerable advances. In 1811, he read a paper before the Geographical Society on the strata in the neighborhood of Bristol, and later, also furnished a publication on the hills in lower Hungary.

In 1812, he returned to Edinburgh where the celebrated Dr. Gregory was his principal teacher in medicine. During the year, he still pursued the study of geology and natural history under Professor Jamieson.

He graduated with twenty-three others on September 13th, 1813, and received the degree of Doctor of Medicine from the University of Edinburgh. His graduation thesis was "De Erysipelate Contagiosa." It is interesting to note that the total output of young doctors from Edinburgh for that year was 62, of whom 28 belonged to Scotland, 15 to England, 9 Ireland, 4 Jamaica, 2 Wales, and one each from Canada, America, St. Croix and the Canary Islands.

Even by this time, Bright gave evidence of the ability that later made him the most distinguished graduate of his year.

Another person, who was also to become famous, was a student at Edinburgh at this time. This was Thomas Addison, who received his degree in 1815, and who was later associated with Bright at Guy's Hospital.

After leaving Edinburgh, Bright decided to graduate also from Cambridge, and, accordingly, he entered Peterhouse, where his brother was a fellow, but after spending two terms he found residence in college incompatible with his other pursuits, and left the University.

He then returned to London, and became a pupil at the public dispensary under Dr. Bateman, who was not only a man of great professional skill and knowledge, but was the chief authority of the day on eruptive diseases. Bright now applied himself closely to the practical part of his profession, losing no opportunity of obtaining information, and looking with particular anxiety to

pathological inquiries. During a considerable portion of this time, he resided in the family of the Rev. Mr. Marsnet, a most respectable and high-minded French Abbé, that he might take advantage of his society to improve his knowledge of the colloquial French idiom.

In the summer of 1814, when the continent became again open to English travellers, we find Bright setting out on another tour. He passed through Belgium and Holland to Berlin, where he spent some months perfecting his French and German, and attending the hospital practice of the leading physicians in the German capital.

From Berlin, he passed to Vienna, where he spent the winter. At that time the famous old Vienna school, among whom were Van Swieten and Auenbrugger, was nearly extinct, and such well known members of the New Vienna school as Semmelweis, Hebra, Skoda and Rokitansky were either in their teens, infancy, or unborn. The sole survivor of the old school was Johann Peter Frank, who, at that time, was the leading medical man in Vienna. Bright thought a good deal of this venerable physician and, doubtless, profited considerably by his association with him. Frank, in our time, enjoys the distinction of being first physician to emphasize the importance of diseases of the spinal cord.

In addition to his medical interests, Bright found time to observe more general matters that were passing before him during the Congress in Vienna at that time. He describes the general condition of science as far from flourishing in that state.

He visited the famous lunatic tower, erected in 1784, which was one of the show places of Vienna. As in ancient Bedlam, here the public were allowed to view the insane, like animals in a menagerie, on payment of a small fee. Bright describes the place as follows: "The asylum is a fanciful edifice and not well contrived. Externally, it appears a large rounded tower, and, on entering, it is found to consist of a hollow circle, in the centre of which, a square building arises, joined to the wall by each of its four corners. The circular part contains the patients, and the enclosed building the keepers. It contained 300 patients, whose condition was far from being as comfortable as many of the establishments for the insane which I have visited."

In March, he extended his journey into Hungary, but returned in the summertime to reach Brussels a fortnight after the Battle of Waterloo. Here, the immense military hospitals, crowded with sufferers, after the great battle, supplied material of professional interest which naturally delayed his homeward journey.

Arriving in London, he resumed his attendance at the public dispensary, where he for some time discharged the duties of assistant physician.

In December, 1816, he was admitted a licentiate of the Royal College of Physicians, and shortly afterwards was elected assistant physician to the London fever hospital, of which Dr. Bateman had hitherto been the sole physician. His public labours were thus rather severe and were varied and increased at Guy's hospital, as often as opportunity would permit.

In the midst of these occupations, he was interrupted for several weeks by an attack of fever, which he caught at the fever home during a very severe epidemic, and narrowly escaped with his life.

It is perhaps due to this unfortunate circumstance, that we are indebted for a most interesting volume, entitled: "Travels from Vienna Through Lower Hungary With Some Remarks on the State of Vienna During the Congress in the year 1814." This was a large quarto volume of 700 pages, and a most valuable contribution on the social condition, statistics and natural history of the country, then so little known in England.

The travels of great physicians have seldom been anything more than matters of casual record, and rarely has a pro-

fessional man left a detailed account of his journeys among strange lands and poples.

It is perhaps not too much to say that Richard Bright's account of his journey from Vienna through lower Hungary is one of the most important books of travel written by a physician, for it is an authentic and scientific account of the state of that kingdom at the time of the peace conference of 1814-1815, just before the close of the Napoleonic wars. Bright's powers of observation and description, his wide reading, his accuracy and his artistic ability, are readily seen by a study of this book. Most of the illustrations are from his own pen.

It has been a matter of comment, that this Hungarian journey was of immense value to Bright in after life, in that the experience increased his faculty of observation, while, in the record itself, he had already perfected his gift of literary expression.

In 1813, he took a second tour through Germany, Italy and France, where he probably came under the influence of Louis and Laennec. In 1820, he finally settled down in London, in Bloomsbury Square, being in the same year elected assistant physician to Guy's Hospital.

In 1822, he became a fellow of the Royal Society. In 1824, he was made full time physician to Guy's, and three years later he published the work, which was to make his name famous to posterity.

To those of us, who have received the knowledge of the connections of dropsy, albuminous urine and disease of the kidney, among the first rudiments of medicine, the facts, which establish that connection, may appear so simple, and so easily ascertained, that the amount of labor, the accuracy of observation, and the rigid adherence to the inductive method, which characterized the whole of Bright's researches, may hardly have been suspected, still less adequately appreciated.

It is strange, indeed, that dropsy should have existed so long, and its cause have been undiscovered, and the renal disease, as we now understand it, should have been almost unknown.

Hippocrates records having observed white clouds in the urine, and, as early as 367 A.D. Aetius associated certain cases of dropsy with diseases of the kidneys. Cotunnuis, in 1770, demonstrated, for the first time, that the urine of dropsical patients could be coagulated by boiling, but, for some time after the beginning of the 19th century, dropsy was regarded, in itself, a primary disease, dependent upon the deficient action of absorbents. The favorite notion of the day, of referring every disease to the liver, was also not without influence, and when morbid anatomy began to be more carefully studied, when the liver was found to be structurally changed, the internal lesions upon which dropsy depended, were supposed to have been satisfactorily demonstrated. But though disease of the liver was a cause in certain cases, in many instances of dropsy, changes in the liver were found not to be present.

A great advance towards the true pathology of dropsy was made by Dr. Blackhall, who, in his treatise on dropsy, in 1913, pointed out that in a great number of cases, the urine could be coagulated upon the application of "heat and nitrous acid," and those, which were for the most part cases of general dropsy, he called inflammatory dropsies. It is, however, remarkable, that although Blackhall availed himself of all opportunities of inspection after death, he never succeeded in connecting the coincidence of these two remarkable phenomena, dropsy and albuminous urine, with disease of any internal organ. Dr. Wells, too, a name scarcely less illustrious, at that time, than Blackhall or Bright, investigated the subject in a most philosophic spirit, but missed, though narrowly, the discovery of the disease of the kidney. Such was the state of knowledge upon the subject, when the inquiry was taken up by Richard Bright.

The first volume of Bright's renowned publication appeared in 1827, just 100

years ago. It was called "Reports of Medical Cases Selected with a Few of Illustrating Symptoms and Cure of Disease by a Reference to Morbid Anatomy." This work was in two volumes, the second appearing in 1830. They contain 65 beautifully coloured plates. They show Bright's faculty of insight, as they are entirely composed of original observations, made at Guy's Hospital, during the six short years he had been on the staff.

The most celebrated part of this book is the first 126 pages of Volume I. This section is entitled, "Cases Illustrative of Some of the Appearances Observable on the Examination of Diseases Terminating in Dropsical Effusion." Bright reminds us that a common cause of dropsy is venous obstruction, but adds that too little attention has been paid to the kidneys. Often albuminous urine, which he detected by heating the urine in a spoon over a candle, is present, as well as dropsy. Then he claims the kidneys will be found diseased.

To support this contention he gives, in great detail, the clinical history and pathological findings of cases, in which albuminous urine was associated with a diseased state in the kidney.

It is to be remembered, that, in those days, physicians had no instruments of precision, except their five senses. Even as late as 1870, the clinical thermometer was considered a curiosity at Guy's Hospital. Bright's economy of means reminds us of Virchow's cutting sections all his days with an old every day razor.

Bright's next allusion to renal disease was in his second Goulstonian lecture delivered in 1833. He refers to the enlargement of the heart, the serious effusions, headache, coma and the cerebral hemorrhage commonly found in association with disease of the kidneys. He concludes as follows: "My conviction is complete as to the existence of some decided connection between the three facts, anasarca, coaguable urine and decreased function going on to diseased structure of the kidney."

His final and most extensive communication on the subject of nephritis, appeared in Guy's Hospital Reports for 1836. It consists of two papers occupying 65 pages. These are masterly descriptions, in which he outlines the signs and symptoms of the disease, and the effect of the retention of urea, in a typical case.

He is not hopeful of treatment, unless the case is seen in the early stages. He states that diaphoretics and hot baths are useful. He advises cupping of the nape of the neck to relieve headache, bleeding is of little value, acupuncture may relieve oedema, leeches to the loins, the only diuretic that may be used is digitalis, elaterium is a suitable purge, milk is one of the best aliments, every precaution should be taken to avoid a chill, and a warm climate is desirable.

Assuredly this is one of the most astonishing series of papers in medical literature. A disease, which presents symptoms of derangement of almost every organ in the body, is unerringly ascribed to the kidney. We are shown how to diagnose the condition, given a description of its cause, symptoms, treatment and postmortem appearance in so complete and accurate a manner, that after a whole century no error has been detected, and, if we accept a few facts that have been gleaned by instruments Bright did not possess, little of importance has been added.

He advanced no special views of pathology, and though from his descriptions, his cases fall into three or four more or less well defined groups, he affixed no patricular labels to his many descriptions, and hence his articles are free from the maze of often meaningless classifications that have arisen and confuse the literature since his time.

Though he made no further publications on renal disease after 1836, his enthusiasm in its study continued, and with the hearty co-operation of his colleagues, two wards were set aside from May to October, 1842, for renal cases only. These were under Bright's care. A small laboratory was established

nearby. This, Bright states, was the first experiment, so far as he knew, that had been made to study a particular disease by bringing patients suffering from it into one ward, properly arranged for observation. The results of this experiment were published in Guy's hospital reports for 1843, and they confirmed Bright's previous investigations.

On account of its immense practical importance, this work soon became widely known. In a short time Morbus Brighti, or Bright's disease, was a familiar appellation over the whole of Europe, and will doubtless preserve the memory of Bright as long as the disease is known by a separate name.

It should be distinctly understood that Bright was not simply a specialist in kidney disease, but a clinical physician of rare excellence, who followed his case to the postmortem room, when unhappily they arrived there, and carefully observed, not only the change which had taken place in the organ whose disease had caused death, but also the state of all the organs of the body.

His work on renal disease has attracted so much attention that too little has been paid to his other observations, many of which were original. His power of seeing was extraordinary, little escaped him, he never saw what was not there, not a single one of his recorded observations have been shown to be incorrect, no preconceived idea tints his descriptions, his industry was unflagging, he willingly went into the country for a postmortem, he could write well, and any disease he describes stands out with all its features clear, and the account he gave is true to-day.

His other studies covered a wide field, epilepsy, tumour of the brain, diseases of the duodenum, jaundice, acute yellow atrophy, peritonitis, and according to Sir Wm. Hale White, Bright must be given the credit of being the first to depict Addison's disease. He did not connect these symptoms with disease of the adrenal, although he described these organs as being greatly enlarged, due to a deposit of scrofulous material.

He was particularly interested in abdominal tumours, and accordingly we find that his last contributions to the literature of his profession were a series of monographs, "Clinical Memoirs on Abdominal Tumours" which, however, want of health and time did not allow him to finish. These were later published by Dr. Barlow in 1860.

In addition to his hospital work, Bright was also a lecturer in the medical school. For three years, he gave a course of lectures on botany in relation to *materia medica*. Later he gave clinical lectures on diagnosis and treatment, for the rest of the time he was active on the staff of the hospital.

Bright's associates at Guy's Hospital were all of them men of strong and striking personality. Such men as Samuel Wells, Astley Cooper, Thomas Addison, Hodgkins and Gull were his contemporaries. Of these, perhaps Bright, Addison and Hodgkin are best known to posterity.

Another worker at Guy's, who is chiefly known as you recall for his description of paralysis agitans, was James Parkinson. Bell, Brodie, Jenne, Lister, Travers, Prudgen and Teale were also prominent figures in English medicine at this time.

In London, in 1839, was published Volume I. of "The Elements of the Practice of Medicine," by Richard Bright and Thomas Addison. The second volume never appeared, although these men were uregd to complete the edition.

In the earlier part of his career, Bright's practice was not large, but, as his reputation grew, he gradually took the position of leading consulting physician in London. In 1832, he became a fellow of the Royal College of Physicians, and in 1837, he was appointed as physician extraordinary to Her Majesty Queen Victoria.

Bright was twice married, first, to the youngest daughter of Dr. Wm. Babington. The only son by this marriage took holy orders, but died young. His second wife was a daughter of Benjamin Follett, of Exeter, and sister of Sir Wm.

Webb Follett. She survived him, as did three sons and two daughters. His eldest son became Master of University College, Oxford. His youngest son was a physician.

Like Harvey, Sydenham, or Jenner, Bright was the typical Saxon physician, and his mind of cheerful cast. Even as a young man, however, he never stepped out of his dignified role to be consciously humourous. There was in his composition not a trace of self-consciousness or whimsicality, nothing of the Celtic habit of saying something funny for its own sake, or that eminently Scotch trait of drawing out other people's peculiarities for private amusement, which was so characteristic of Dr. Wm. Hunter.

He was by general admission a man of fine and attractive nature, and from early manhood he was animated by a genuine love of the truth and unswerving sense of duty. He was of an affectionate disposition, and loved his home and family life.

He was a well balanced, solidly built man of medium height and of a remarkably firm, even, and self-contained character. He was widely accomplished, a good linguist, well versed in more than one science, a creditable amateur artist, and possessed of much taste in art. He was well cultivated on all sides by travel and society.

In his intellectual character, the first feature which strikes one is a certain simplicity. Beyond most observers, he succeeded in viewing objects without prejudice. Not putting forward any theory himself, he was not trammeled by any of the prevailing systems of medicine. He had a remarkable ability of picking out the important facts in any subject and combining them to explain each other.

He was not generally regarded as a brilliant man. He had little power of exposition and in his own school, while his fame was rapidly spreading over the whole civilized world, he was less popular and impressive, as a teacher, than his brilliant colleague, Thomas Addison, though the latter was much less known to the outside public.

All his contemporaries agree that truth and honour, generosity and fair play were the essence of Bright's nature, and in the record of his travels and in the story of his life, we do not find a trace of that over estimation of self which we call conceit, the hallmark of intellectual dishonesty. His charcter reminds us of the truth of Emerson's aphorism that "Great men do not live for show." He was somewhat oblivious of the importance of his own work and incapable of anxiety about its future fortunes. His scientific work, like Addison's, reaches far out into the medicine of the future, and in the hundreds of pathological reports, which he has so carefully recorded, it is probable that the physicians of to-morrow will find correlations, as yet unthought of.

He died at his home, 11 Saville Row, on December 16, 1858, after a very short illness. A postmortem was performed, which disclosed the cause of death as being in the aortic valves. They were replaced by a ring of bony matter, which closed the artery entirely, except for a small longitudinal slit, the edges of which were perfectly rigid for about two-thirds of their extent.

Dr. Bright had advanced far into the vale of years, but not without frequent warnings of internal decay. His symptoms were very dubious. Although he would place himself under medical treatment, he had always an unsuperable objection to allow a personal examination. His death occurred suddenly after vomiting blood.

He was buried at Hensal Green Cemetery, and a mural monument was erected to his memory, in St. James' Church, Piccadilly. The Royal College of Physicians possess his portrait in oils and also a marble bust. Another bust is located at Guy's Hospital.

The *Lancet* of December 25, 1858, in writing of Bright's death says: "The sudden and unexpected demise of Dr. Bright has created a deep impression of

grief and regret, such as only a sense of irretrievable loss could occasion. In him, all feel that the medical profession of England has lost one of the most original, observant and philosophic minds. A man of peculiar independence of thought, of high morale and untiring energy he contributed more perhaps than any other to mould the medical opinion of his day. By his singular devotion to pathological investigation which characterized his career he was at once enabled to accomplish a work which has immortalized his name.''

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Protein Asthma: Nature and Diagnosis.

(H. K. Detweiler, M.D.)

The following simple etiological classification o fasthma is suggested as a basis for this discussion:

1. Protein asthma.
2. Bronchitic asthma.
3. Cardiac asthma.

PROTEIN ASTHMA: In 1906 Wolff-Eisner suggested the anaphylactic nature of the condition known clinically as hay fever. The relation of certain proteins, particularly those of pollens, to this disease seemed fairly obvious, and the method of demonstrating the sensitivity of the patient was so simple that it was not long before the theory was accepted generally. In 1910 Meltzer suggested that asthma might also be a phenomenon of anaphylaxis. He based his theory upon certain facts elicited in the study of anaphylaxis by Auer and Lewis working in his laboratory at the Rockefeller Institute. These facts may be enumerated as follows:

(a) In an anaphylactic attack the bronchi are constricted.

(b) The constriction is of peripheral and not of central origin.

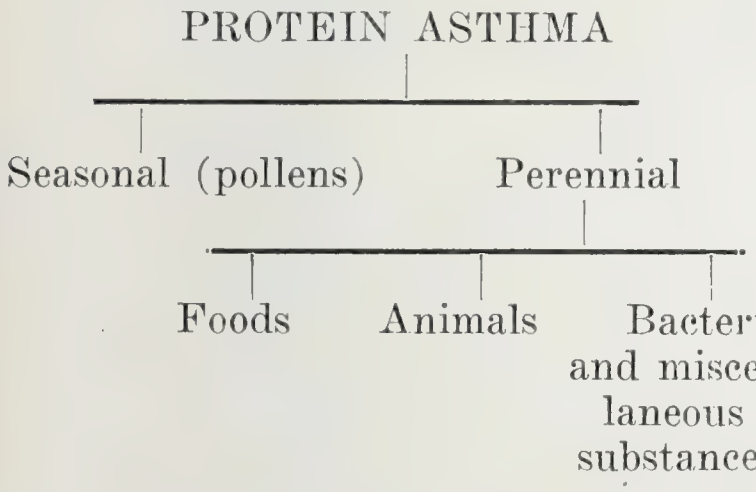
(c) Atropine relieves the anaphylactic attack.

He argued that these conditions undoubtedly obtain in asthma. He pointed out that they obviously were present in the asthma associated with hay fever, and it was his belief that they would hold for other forms of the affection.

Meltzer's theory was put to an exhaustive clinical test during the next few years, and while it has not been proved to hold in all cases of asthma, it has been amply substantiated in a large

group of cases. Indeed it has served sharply to divide all cases into two great sub-divisions: (1) those which are sensitive to protein; and (2) those which are not. The former are now regarded by careful observers as comprising the whole group of primary asthmatics, while the latter, or non-sensitive cases comprise a mixed group, composed chiefly of cases of asthma in which the primary factor is an infection.

Protein asthma may be subdivided into two main types on a basis of seasonal and non-seasonal incidence; the following table adapted from Walker will illustrate this:



In an uncomplicated case of protein asthma the clinical picture of a severe attack would be as follows:

Upon exposure to the protein to which the patient is sensitive, there is usually a feeling of tightness in the chest, which rapidly grows more and more intense until the breathing becomes little more than a succession of quick gasps, followed by prolonged and wheezy expirations. The main difficulty seems to be in expiring the air, with the result that although all the muscles of respiration are brought into play, the expiratory act is so long that the impulses to inspire comes before the chest has emptied itself. Thus each succeeding respiratory cycle leaves the chest with an additional amount of residual air. The net effect is that it is not long before the lungs are greatly distended, the diaphragm is low, the superficial cardiac dullness obliterated, and the whole chest is being held in an extreme inspiratory position. Fortunately, just as it would seem that the patient must suffocate, there comes a tendency to cough and a thick, tenacious, jelly-like material is brought up in increasing quantities. With the advent of this sputum, or probably just preceding it, the bronchial spasm begins to lessen and in a comparatively short time the attack has subsided. The whole cycle may last from half an hour to three or four hours, and there may be a succession of attacks with little respite in the intervals. Between the attacks, physical examination of the chest reveals no abnormality whatsoever, but during the attack, in additional to the signs already enumerated, there are to be noted hyperresonance, prolonged expiratory murmur and numerous dry rales, mostly of high and medium pitch.

If at the height of an attack the patient is given a hypodermic injection of a few minims of adrenalin chloride 1-1000, relief is experienced in a few moments. Atropine has a similar effect, but the action is less rapid. It is generally assumed that the adrenalin acts upon the sympathetic nerve-endings causing a relaxation of the spasm of the bronchial musculature, and this relaxation has the effect of restoring the lumen of the bronchus concerned to its normal size. This view is supported by the findings in sensitized animals who have been given a second dose of protein with the production of anaphylactic shock. If the animals are allowed to die in anaphylaxis, post mortem examination reveals a marked spasm of the bronchial muscles. If, on the other hand, the dose of protein is not too large, and the adrenalin is administered, the anaphylactic condition is relieved and the respiratory embarrassment of the animal ceases in a manner similar to that of the human asthmatic.

Many observers believe, however, that the respiratory difficulty in human cases of asthma is not due to bronchial muscular spasm, but rather to the turgescence of the mucous membrane of the bronchial tree as a local effect of the protein to which the patient is sensitive. This turgescence of the mucous membrane is

claimed to be not unlike the local reaction of the skin upon the application of such protein to abraded areas. The proponents of this theory claim that the action of adrenalin in these cases is similar to its action in urticaria; that is, it acts upon the smooth muscle of the smaller blood vessels and on the contractile cells of the capillaries.

DIAGNOSIS: It is well here to emphasize the importance of a careful history. According to Walker "four-fifths of the cases beginning their asthma in infancy are sensitive. Two-thirds of those beginning in childhood are sensitive, while only one-half of those beginning in young adult life, and one-third in full adult life give a positive test. When the asthma begins after the age of forty-five, practically none are sensitive. From this it is clearly evident that the history of onset aids materially in the diagnosis.

We have found that an accurate account of the first attacks, particularly with regard to whether asthma or bronchitis appeared primarily, is also of great value, especially in the patients with an onset after twenty years of age. In those with earlier onset, it is doubtful if the best clinical history can render unnecessary the carrying out of the protein tests to be described.

The physical examination of cases of uncomplicated protein asthma shows the chest held in the inspiratory position with diminishing excursion deeper resonance, prolonged expiratory murmur and numerous dry râles. The breath sounds are frequently difficult to hear, partly by reason of the adventitious sounds which usually are very prominent, and partly by reason of the lessened air entry. Often this suppression of the breath sounds is heard more particularly over certain areas, suggesting, therefore, that the bronchial spasm or turgescence may be irregularly distributed, or that it has actually plugged the bronchus supplying the area referred to. Between attacks, in the uncomplicated cases, there is no abnormality to be found on physical examination of the chest. On the other hand, if the patient has been a sufferer from asthma for a considerable time, chronic pulmonary emphysema supervenes, accompanied by secondary bronchitis. In such cases, examination between attacks reveals the characteristic physical signs of this condition. At this stage the protein asthmatic resembles the patient with bronchitic asthma, and a differentiation becomes impossible from physical examination alone.

In the uncomplicated protein asthma cases the sputum is usually clear and jelly-like, and is only present during an attack, or for a few hours after. It therefore differs in its character from the sputum in the cases of bronchitic asthma, in which there is usually a yellowish or greenish type of sputum; in other words the sputum is of a purulent character from a pyogenic infection.

Finally, cases of protein asthma will show in nearly every instance a response to one or more of the cutaneous protein tests. A brief description of the technique of these tests follows: A sufficient number of small abrosions are made upon the ventral surface of the forearm to accommodate the various proteins to be used. These scratches may be made with a sharp scalpel and should be about one inch apart, an eighth of an inch long, and need not be deep enough to draw blood. A drop of N/15 NaOH is placed upon each abrasion, and a minute amount of the protein powder rubbed into it. The first scratch nearest the bend of the elbow is left without protein to serve as a control, because it is occasionally found that the mechanical scratching of a sensitive skin will cause a small wheal to form. The proteins must be kept in solution by replenishing the soda solution as it evaporates, and one-half hour should elapse before the test is complete, and the proteins removed.

In the average case of true protein asthma, it will be noticed within a few

minutes that a red areola is appearing about one or more of the abrasions to which offending proteins have been applied. In the centre of this areola, about the scratch, a wheal will be observed, which gradually increases in size until its maximum is reached within thirty minutes. The smallest wheal which is considered positive, and therefore of diagnostic importance, is 0.5 cm. in diameter, in the absence of any reaction at the site of the control abrasion, may be considered as suspicious, and worthy of further consideration.

The routine series of proteins for cutaneous testing should include the following representatives: *Pollens*: June grass, redtop, timothy, orchard grass, ragwood, goldenrod and daisy; *Miscellaneous substances*: orris root and house dust; *Animal emanations*: horse dander, cat hair, cattle hair, dog hair, sheep wool, rabbit hair, chicken, goose and duck feathers. Of the foods the following should be represented: *Cereals*: wheat, oats, corn, rice and rye; *Meats*: beef, pork, lamb and chicken; *Fish*: salmon, mackerel, lake trout and white fish; *Vegetables*: potato, cabbage, spinach, cauliflower, onion, turnip, parsnip, lettuce and celery; *Fruits*: orange, tomato, apple, peach, prune and pineapple; *Miscellaneous foods*: milk and eggs.

Of the cases of seasonable asthma the large majority react to one or more pollens, and their symptoms of asthma are usually associated with those of hay fever. Of the non-seasonal cases of asthma the animal emanations appear to be the most frequent cause in adults, whereas foods, particularly eggs and milk, are the most frequent offenders in children.

In connection with dust it is important to realize that it may act in two ways. First, by mere mechanical irritation: most asthmatics volunteer the information that inhalation of dust of any kind is apt to make them worse. On the other hand there are those who react to the protein contained in house dust extract. It is sometimes remarkable to note the specificity in these cases; certain individuals will react to the dust of a certain house and not to that of any other. In my experience, however, the majority of dust sensitive cases will have an attack wherever house dust is plentiful. The following case illustrates this type: C.A.S., a commercial traveller, aged 42, consulted me in January, 1925, because of asthma, to which he was subject throughout the year. He was unable to offer any clue as to the causation. The test with stock house dust extract yielded a violent reaction. After discussing this feature of his case with him I was able to elicit the following interesting history: He frequently had attacks of asthma during the week days while on the road, but was always free, or nearly free, from attacks while at home over the week-end. He always had severe attacks on visiting his mother. The explanation of this fact was that his wife was a very scrupulous house-keeper, so much so that he confessed he felt that she was a slave to her housekeeping. His mother was getting on in years and no longer supervised the keeping of her house, and the servants allowed a great deal of dust to accumulate.

There are occasional cases in which the patient exhibits asthmatic attacks upon the ingestion of certain drugs. The commonest of these drugs are aspirin and quinine. Sometimes very severe reactions occur when these drugs are taken, and it is usually unwise to disregard a patient's statement that he thinks asthma is brought on by the taking of any given drug. I have seen several aspirin cases which had had severe attacks of asthma, but these passed off without serious results. However, alarming cases are on record, such as Kern's patient, who mentioned to her former physician that she was not able to take quinine. Curiosity, however, led him to give her a mixture containing $\frac{1}{8}$ of a grain of quinine to the dram. Within a few minutes after the first dose the patient almost died of

angioneurotic oedema with oedema of the glottis.

The points brought out in the diagnosis and differential diagnosis of protein asthma may be summed up in the following table:

TABLE OF DIFFERENTIATION.

Protein Asthma.

History—Usually early onset.
No bronchitis until later in the disease.
Clear jelly-like sputum.
Clinical Exam.—Expiratory difficulty.
Chest clear between attacks.
Laboratory tests.—Protein tests positive.

Bronchitic Asthma.

Usually late onset.
Asthma follows on a history of bronchitis.
Usually muco-purulent sputum.
Inspiratory difficulty.
Signs of bronchitis between attacks of asthma.
All protein tests negative.

This paper is concluded in the next issue by a discussion of bronchitic asthma an dtreatment of asthma.

The Induction of Labour—Its Indications

G. B. White, B.A. (Cantab.), M.B.

THE term Induction of Labour is here taken to mean the production of uterine pains and contractions resulting in the termination of pregnancy, at any period after the sixth month of pregnancy.

Those who favour the induction of labour in certain cases have been accused of "meddlesome midwifery." Wright, of Toronto, has answered this charge with the statement: "If we can perform this operation in such a way that it causes no danger, or at least very much less danger to the patient than the prolongation of pregnancy involves, then we must conclude that some interference is not only justifiable but advisable."

Indications.

The function of the obstetrician is to bring as many as possible of the pregnancies which come under his care, to a successful conclusion both as regards the life of the child and the life and health of the mother. Induction of labour, then, is indicated only when it is believed that the continuation of the pregnancy will endanger the life of either the child or the mother.

Indications When Child's Life Is Endangered.

The prolongation of pregnancy may endanger the child's life when it is allowed to proceed to such a point that the child is relatively too large compared with the mother's pelvis. Such disproportion may exist when the pelvis is contracted, or when, with a normal pelvis, the foetus is allowed to advance to postmaturity.

Induction of premature labour may be considered advisable when the amount of pelvic contraction present is such that it will not allow the foetus to pass through the pelvis at term, but will at an earlier date allow of the birth of a child which, with average care, can be reared. Hess has pointed out the high mortality among premature infants[1] and induction of premature labour for contracted pelvis was objected to on these grounds, but our more recent increase in knowledge of the care of premature infants makes the risk now almost negligible. Ivens, Cantrell and Reid conclude that by induction of labour in suitable cases, a considerable amount of unnecessary

suffering from prolonged labour due to minor pelvic contraction may be avoided. In addition, induction of premature labour is not as inimical to the child as published statistics maintain.[2] In the interests of the child, labour should be induced as near to term as possible, but before disproportion becomes apparent. It should never be attempted before the 34th week and preferably not before the 36th week. It should never be attempted where the true conjugate is 8 cm. or less. Bailey states that induction of labour may be employed in primigravidae with some slight degree of pelvic contraction, where the presentation and position are normal and the head is either movable at the pelvic brim, or fixed at the pelvic brim but with some very slight degree of "overlapping," or fixed but high, it being considered that delivery by the natural route is impossible.[4] It may also be employed in multigravidae, with or without pelvic contraction, where there is a history of previous difficult labour or habitually postmature foetus.[5]

The alternative of induction of premature labour for contracted pelvis is Caesarean section at term, which is claimeed to be a simple operation and more certain in results. It is essential where the true conjugate is less than 8 cm., whether the pelvis is flat or generally contracted. Williams claims that induction of labour is in no way justifiable, since, in cases of moderate pelvic contraction, equally good results for the mother and far better results for the child will be obtained by subjecting the patient to a test of the second stage of labour and resorting to Caesarean section should nature prove inefficient. At the Johns Hopkins Hospital they reject on principle the induction of premature labour for contracted pelvis.

In considering postmaturity, it must be remembered that a pregnancy protracted beyond the calculated date is not evidence that the child is postmature, nor is it, in itself, an indication for induction. But any such pregnancy calls for close observation of the relationship between the size of the foetal head and the mother's pelvis, with particular attention to primigravidae, especially elderly. If any disproportion is threatened with the growth of the foetus, pregnancy should be terminated at once in the interests of both mother and child, even though the pregnancy is but little prolonged.

Early termination of the pregnancy is indicated where there is an extreme degree of obstruction and Caesarean section is refused. Such cases are:

Extreme pelvic contraction,
Advanced carcinoma of the cervix,
Other irremovable malignant tumours, as of the pelvic bone.

Indications When Mother's Welfare Is Endangered.

The mother's life or health may be threatened by conditions due directly or indirectly to the pregnancy, or by pre-existing diseases aggravated by the pregnancy. The former group may be subdivided into:

(a) Toxic group,
(b) Haemorrhage group,
(c) Other conditions.

The *toxic group* includes:

Eclampsia and toxaemic albuminuria.
Chorea gravidarum,
Habitual death of the foetus.

In these conditions, it should be attempted to clear up the disease and prolong the pregnancy as long as possible.

Hyperemesis gravidarum should be treated medically whenever possible, but if the "Water Treatment" fails to produce diuresis and clinical improvement within six days, then the pregnancy should be terminated. Termination of the pregnancy is also indicated when there are grave clinical signs, as a rising pulse with falling blood pressure, fever, jaundice with liver shrinkage, and acetonuria. It is better to

err on the side of too great haste in terminating the pregnancy than to allow it to proceed until it is too late.

In pre-eclamptic conditions, one should not have too conservative as regards the life of the child. The object should be, not to bring the child as near term as possible, but to the period of viability. Medical treatment should first be tried, but where treatment brings no improvement, then the pregnancy should be terminated before actual eclampsia. Induction should be considered when there is persistence of any or all of the following signs:

High and increasing blood pressure (180 or over),
Failing vision,
Persistent headache.

With the advent of actual eclampsia, two courses of treatment are available. The object of the first, or radical treatment, is to terminate the pregnancy immediately. With this, the maternal mortality is 35 % and the results are not good compared with those of conservative treatment, in which the convulsions are treated and the pregnancy is disregarded; if labour does not supervene, then it may be induced when the convulsive crisis is over. With this treatment, the maternal mortality is 10%. In these cases, is Caesarean section is ever justifiable, it is in elderly primiparae or where labour promises to be exceedingly difficult or prolonged.

In cholera gravidarum where the condition has not been amenable to general treatment, induction of labour has been advocated by some. It seems doubtful, however, whether this gives any marked improvement, and Ley states that without exception it is attended by bad results. Moreover, Fehling gives the maternal mortality as 36% when induction is attempted.

Where there has been habitual death of the foetus, by taking a careful history of previous pregnancies, it may be calculated when the pregnancy may be expected to end. Induction of labour may then be performed one week before the miscarriage is due.

The *haemorrhage group* includes:

1. Threatened abortion.
2. Accidental haemorrhage.
3. Placenta praevia.
4. Hydatidiform mole.

In threatened abortion, preventive treatment should cease and the pregnancy be terminated when the haemorrhage persists and is abundant enough to produce constitutional effects, such as anaemia, feebleness, rise of pulse rate, etc.

In accidental haemorrhage, the foetal morality is always high, so that prime consideration should be given to the life of the mother. Many writers advocate the immediate emptying of the uterus in all cases, but this should not be done in the milder forms where the loss of blood has not been great enough to cause any systemic symptoms, where there is no appreciable change in the character of the foetal heart sounds, and where the signs of bleeding have ceased, whether visible or concealed.

In placenta praevia, also, the mother's life should receive the first consideration and the pregnancy should be immediately terminated. Where the placenta is marginal or lateral, induction may be employed, but where it is central, Caesarean section must be resorted to.

In the case of hydatidiform mole, there is a tendency towards spontaneous expulsion of the mole, but this is seldom complete, so that steps should be taken to empty the uterus as soon as this condition is diagnosed.

Other conditions due to the pregnancy where induction may be necessary, are:

Hyddramnios,
Retention of dead ovum,
Infection of uterus,
Irreducible prolapse of gravid uterus.

Induction of labour is always indicated in acute hydramnios owing to the great discomfort resulting and the danger to the life of the mother as a result of the sudden spontaneous rupture of the membranes. Little consideration need be given to the child, since 50% are monstrosities and many of the remaining 50% are too poorly developed to have a chance of life. In chronic hydramnios, induction may be indicated when there are severe pressure symptoms.

General diseases which may complicate pregnancy and necessitate termination of the pregnancy, are:

Uterine leiomyomata,
Cardiac disease,
Advanced pulmonary tuberculosis,
Diabetes,
Pyelitis,
Acute and chronic nephritis,
Pernicious Anaemia,
Acute Yellow Atrophy,
Exophthalmic goiture,
Insanity.

Induction of labor is seldom indicated when a pregnancy is complicated by fibroids. Fibroids in the lower uterine segment or cervix, though not interfering with the pregnancy, may cause mechanical hindrance to labour. In such cases, rather than induce premature labour, the pregnancy should be allowed to go to term and the child be delivered then by Caesarean section. When fibroids are present in the body of the uterus, there is a tendency to abortion. In these cases spontaneous abortion is of less danger to the patient than interference, which should not be attempted. In all fibroids complicated by pregnancy there is the risk of degeneration, especially necrobiosis. Such cases should be carefully watched for signs of degeneration and if these appear, the tumour or whole uterus should be removed, without regard for the child, but induction is not indicated.

In patients suffering from cardiac disease, it is always difficult to determine to what extent the heart must be damaged before it is no longer competent to endure the strain of labour. Bierring quotes a series of 87 women suffering from heart disease who experienced 107 labours, producing 98 living children; only 11 of the women developed severe heart symptoms and the mortality was 6, all of the melderly multipara. 50% showed no symptoms throughout the pregnancy.

This seems to point to the fact that pregnancy has not nearly such a harmful effect upon a previously existing heart lesion as would be expected. If, however, the more remote effect of the pregnancy were considered, it would probably be found to be not so favourable. Prassulides states that danger comes with complications, and that of all defects, mitral stenosis is the most inclined to become aggravated. Sir James Mackenzie states that the pregnancy should be terminated: (1) when orthopnoea has been reached. (2) in mitral stenosis, when oedema of the lungs tends to persist in spite of sitting up in bed or when the heart rate is persistently over 100 per min. with violent palpitation on the slightest exertion. (3) in aortic regurgitation with a Corrigan pulse or with a distinct forcible apex beat outside the nipple line. (That is, in almost all cases of aortic regurgitation).

D. G. Campbell concludes that:

1. Auricular fibrillation, myocarditis and mitral stenosis are very adversely affected by pregnancy.

2. In women with these conditions, the pregnancy should be aborted and the patient sterilised without waiting for signs of breakdown.

3. If there is mitral stenosis without evidence of myocarditis, the patient is capable of bearing one or two children safely.

4. Induction or Caesarean section is indicated with dyspnoea, palpitation, precordial pain and rales at the bases of the lungs.

5. Abdominal Caesarean section is not as satisfactory where there is car-

diac failure, as induction of labour or vaginal Caesarean section.

6. The condition of the heart muscle is the factor of importance.

G.B. Smith states that those who would survive a prematurely induced labour would pass equally well through labour at full term and those who appear likely to die at labour are equally likely to die as a result of premature induction; he therefore favours Caesarian section at term. This has been performed by many others with very satisfactory results so that in the majority of cases Caesarean section appears to be more favourable than premature induction of labour. It must, however, be remembered that it is not only the actual labour that will throw an extra strain upon the heart, but that the pregnancy itself will often cause a very great increase of cardiac distress necessitating induction of labour very early in the pregnancy, and this should be done before the signs of breakdown appear.

Induction of labour is not often beneficial for pulmonary tuberculosis, but it may be indicated with complications or where the condition is very advanced, endangering the life of the mother. Nearly 100 years ago, d'Outrepont stated that pregnancy should be determined when the condition of the mother was so serious as to make it improbable that she would live until term, and this still holds good.

A condition of specific renal glycosuria with low threshold but normal blood curve, frequently occurs in pregnancy. If the glycosuria is not renal, then the patient is either a diabetic who has become pregnant or a pregnant woman who has become diabetic. A patient who is pregnant and has diabetes should be allowed to go to term if the disease can be satisfactorily controlled either with insulin or by dietetic treatment. If the patient cannot be kept sugar-free, the pregnancy should be terminated.

Induction of labour is indicated in pyelitis only if it passes into the more serious condition of pyelonephrosis. It may be used in occasional cases of Pernicious Anaemia when danger of life is threatened. There is no evidence that the termination of a pregnancy is beneficial in this condition.

Acute Yellow Atrophy occurs in women in 75% of cases, and of these, 50% are associated with pregnancy. The condition is sudden in onset, lasts only a few days and is always fatal. With the onset of vomiting and diarrhoea, extreme jaundice and all the indications of dehydration, the pregnancy should be terminated immediately, either by Caesarean section in skilled hands or by induction of labour by the most rapid method.

The concluding section of this article in next issue contains methods of induction and a biography.

THE QUECKENSTEDT TEST.

This test by its simplicity and great practical value merits universal application in diagnosis of compression of the spinal cord.

By compressing both jugular veins the brain becomes congested and enlarges causing an increase in intracranial and intraspinal pressure.

The technique is simple. A lumbar puncture is done in the usual way and pressure manoeter attached. Both jugular veins are now digitally compressed 1½'' below the angles of the jaw.

If there is no obstruction of the spinal canal the pressure rises immediately; with partial obstruction the rise is gradual. There is no rise in pressure if the block is complete.

This test is of immense value in such conditions as spinal cord tumours, spinal cord compression complicating Pott's disease, abscess of the spinal canal, spinal injuries, transverse myelitis, syringomyelia, lateral sclerosis, posterolateral sclerosis, etc.—(Abstract from Piedmont Hospital Bulletin, April, 1927.)

Education of Deaf and Deafened Children.

BY IMOGEN B. PALEN.

JEROME CARDAN, an eminent Italian physician and mathematician (1501 — 1576) may be called the Christopher Columbus of education for the deaf. To him is credited the first authoritative published statement that a deaf-mute child may be taught to read and write. Until then, all down the centuries of social and cultural progress in the old world, educationalists and law-makers alike had classed deaf-mutes with idiots. It is said that the early Church, arguing from the premise that "faith comes by hearing," believed them damned. Justinian embodied in the Corpus Juris Civilis a decree that no deaf-mute could make a will. Lucretius, in his poem on "The Nature of Things," says:

"To instruct the deaf no art could ever reach,
No care improve them, and no wisdom teach!"

The most notable of the hardy adventurers who began to explore the new world of education, after Cardan's discovery, was Pedro Ponce de Leon (1520—1594). He was a scion of a noble Spanish family. In later life he became a Benedictine monk and in a legal document, whereby he donated funds for a chapel, he states that the money was derived in part "from pupils, who were deaf and dumb from birth, whom I have taught to speak, read, write and reckon . . . some of them also to learn Latin and some both Latin and Greek."

The first book published in the English language advocating the teaching of speech and speech-reading to deaf-mute children was written by an English physician, Dr. John Bulwer, in 1648, under the title of "Philocophus, or the Deafe and Dumbe Man's Friend." Dr. Bulwer believed himself to be the discoverer of the "terra incognita of ocular audition," to use his own description. He even proposed to found an academy for the deaf and dumb. He found little encouragement among his confreres, however, who (he writes) "tooke the tearms and expressions this Art justly usurpes for insufferable violation of their reason."

In 1692 Dr. John Conrad Amman, a noted Swiss physician, published a treatise on "how to teach one born deaf to speak." This created somewhat of a sensation and brought many requests for further amplification of the subject. He then brought out a larger volume, "Surdus Loquens Sive Dissertatio Loquens," written originally in Latin and afterwards translated into several languages.

Among some of the earlier oral educators of the deaf there seems to have been an inclination to keep their methods a secret. Samuel Heinicke (1727—1790), a German scholar and musician, founded an oral school for the deaf at Leipsig which marked a new era in the education of the deaf in that country. In a letterto Abbe de l'Epee he writes "the method I now use was never known before and I am not inclined to let others benefit from it for nothing."

Abbe de l'Epée was a self-sacrificing French priest, who devoted his life to the deaf. He is famous as the inventor of the sign-language. When he began his work among deaf-mutes he taught them orally. Soon, however, they began to come in such numbers that he could not possibly teach them all. In his desire to give some help to all of them, he invented the sign-language in order to enable himself to cover a wider field in his ministrations.

In 1895 the United States Government sent a young teacher named Galladet to England to learn how to teach the deaf orally, with a view to provid-

ing for their education. Mr. Gallaudet, on arriving in England, went to a family of the name of Braidwood, who were teaching deaf-mute children orally where he learned to his dismay that in order to learn their methods he would have to bind himself to remain with them in training for seven years and to pay a large fee for his tuition. He thereupon went to France and sought assistance of Abbe de l'Epee, which was freely given. As a result, the sign-language was introduced into the United States and established as the method of instruction in the first state school for the deaf on this continent founded at Hartford in 1817. For the next fifty years deaf-mute children were taught by means of the sign-language and became a class using a language of their own, cut off from their fellows in the community life of ordinary speech.

Alexander Graham Bell was the first outstanding advocate, on this continent, of the oral method of teaching the deaf. His invention of the telephone was the outcome of his endeavours to find a means of transforming sound vibrations into light vibrations in order to make speech "visible" to the deaf. He was the founder of the American Association to Promote the Teaching of Speech to the Deaf. Oralism began gradually to displace manualism. Today, the oral method is used in nearly all state and provincial residential schools for the deaf and in all day-classes for the deaf in the public schools and graduates of such "pure oral" schools as Clark School, Northampton, Mass., are now entering universities where they compete on equal terms with their hearing class-mates.

The road to this desired goal, however, is a difficult and discouraging one. In mental development deaf-mute children are several years behind hearing children of the same age when they enter school at six or seven years of age. This is due to the fact that they have had no exact language of speech in which to form and express their thoughts. Starting with this handicap, they must then be taught speech element by element, syllable by syllable, word by word, with their eyes on the teacher's lips and their fingertips on her cheek. Thus, very slowly, through the medium of their visual and tactile senses only, they learn to imitate the correct articulation of the speech organs and the proper moulding of the vocalized breath for the production of speech.

The speech of deaf-mute children lacks nicety of inflection, tone quality and pitch. They have no auditory sense to monitor their efforts at speech, as a hearing child has, and spoken language is not native to them; like foreigners, they speak it "out of tune." In our schools there are many children of foreign speech who may never be able to master all the niceties of rhythm, idiom and pronunciation of English speech, but no one would think of saying that they should not be taught to speak English, but merely to write it, and that they should communicate with others by means of writing and gestures only. Similarly advocates of oral teaching for the deaf argue that all deaf-mute children should be taught to speak, although their speech may never attain perfection, so that they may not be limited to writing and gestures in their intercourse with the speaking community in which they live.

There is no royal road to teaching speech to the deaf, but the public seem to think that the teacher possesses some mysterious and magic power and should be able to invoke it to produce perfectly modulated voices in children who have never heard, and can never hear a human voice. This arises from the old belief that speech is inherent and from lack of a realization of the fact that speech cannot be developed except by the exercise of certain muscles and by imitation. For months before a hearing child utters his first word, his brain has been a storehouse of countless verbal images. A congenitally deaf child has no such resources from which to draw language.

We who have taught the deaf believe and earnestly hope that science will sooner or later reveal a secret which will enable the deaf to intelligently perceive the modulations of the teacher's voice. At the Convention of Progressive Oral Advocates, held in New York City in June last, a demonstration was given by a blind-deaf boy, Ted Chapman, who could comprehend spoken language not only by placing his fingers on the speaker's lips, chin or the nape of the neck (as is done by Helen Keller, Orma Simpson and others), but also and quite as easily by holding the palm of his hand at a distance of six inches or more from the speaker's face. This boy's teacher began by training the tactile sense along the usual methods employed by teachers of the deaf and did not begin with finger-spelling as in the case of Helen Keller. Dr. Max Goldstein, Superintendent of Central Institute for the Deaf (St. Louis) and editor of "The Laryngoscope," said, in commenting on this demonstration, that it is probably the greatest pedagogical achievement of all time. At the same Convention there was a demonstration of the Osiso by its inventor. It is an instrument for the translation of sound waves into light waves. Each vowel sound spoken into the instrument gave its own unique and characteristic projection of light and photographs of these projections were taken. We hope that in the not too distant future some machine along this line may be perfected whereby it will be possible to present to a deaf child an actual visualization of his own speech and of his teacher's, so that he may compare his efforts at speech with the speech of normal-hearing persons and may learn to interpret the visual characters of speech with as little difficulty as in learning to read the printed page, which we all must master, but which is not a true picture of the spoken words despite its phonetic qualities.

So much for the achievements of the past and the hope of the future—but what of the present? What can the family physician tell the mother of a born-deaf child to lessen her horror of that word "deaf-mute" and what advice can he give to the mother of a child whose acuity of hearing has been seriously impaired by some infectious disease or other cause? In both cases, *tell the mother to treat the child as though he could hear perfectly and to insist that he comprehend the spoken word by looking at the speaker's face.* The pity is that the mother, realizing that her child cannot hear her, no longer tries to teach him speech as she does with her other children, but resorts to gestures and signs. Even the baby deaf can be taught to lip-read at least a few essential words if they are repeated hundreds of times while he is watching his mother's lips and if the comprehension of these words leads to the gratifying of his desires. The mother should be unceasing, day by day, in teaching her child to comprehend speech. When he is old enough to go to school (five years for the day classes in Toronto, six years for the Ontario School for the Deaf at Belleville) he should be sent without delay. Each day of the time in which he is to acquire his education is very precious. He will be taught to speak by specially trained teachers, known as oral teachers of the deaf. At the end of his first year he will have learned to speak, lip-read and write from 50 to 100 nouns, 20 to 30 commands and a few adjectives, and will be able to form and speak simple sentences from his acquired vocabulary. Perhaps it will be five or six years before he can talk at all well, but in the meantime he will have been learning arithmetic, geography, history, reading, etc., and should, if of normal mentality, be able eventually to enter high school or college. This is the picture to hold before every mother of a born-deaf child or of a child who becomes deaf before he has learned to speak.

In addition to the provincial School for the Deaf at Belleville, there are

three oral day classes for the totally deaf at Clinton Street School in Toronto. The establishment of such classes in large cities throughout the continent is of comparatively recent development. Residential schools for the deaf have for very many years provided instruction in trades and manual arts for their pupils, so that the children have initial training for earning a livelihood when they leave school. Now that manual training, leading up to technical high schools, has been introduced into our public schools the troduced into our public schools the pupils in the oral day classes for the deaf may receive the benefit of that training as well as the regular academic instruction.

For very hard-of-hearing children, of normal or nearly normal speech, there are two special classes in Toronto —one in Rose Avenue School and one in Dovercourt School. These are small classes under specially qualified teachers. Pupils eligible for these classes are those who are already retarded on account of impaired hearing, those who are so deaf that they cannot acquire new language forms even when given a front seat, those who are suffering from a nervous strain caused by their efforts to hear and those who have speech defects due to loss of hearing acuity in early childhood.

For the slightly hard-of-hearing who are in danger of being retarded, there are visiting teachers of lip-reading. Dr. Frank W. Bock, Director of the Deafness Prevention Clinic, Rochester, N.Y., says that children who have one good ear and one bad ear "have lost their margin of safety." He advises instruction in lip-reading even when the child can hear fairly well with the good ear. In Toronto the visiting teachers go to four schools a day, giving two or three one-hour lessons at each school each week.

In all three branches of this work in our schools (oral day classes for the congenitally deaf, special classes for the very hard-of-hearing and the visiting lip-reading work) a great deal of attention is paid to the development of residual hearing.

In Toronto there is a very active Lip-Reading Club for Adults (Secretary, Miss Alice Hetherington, 112 Hilton Avenue) which meets twice a week. Notices of the meetings appear in the daily papers.

Behind all this work lies the thought expressed in the slogan of the Federation of Leagues of Hard of Hearing—"The Passing of Deafness!" The United States Government Bureau of Statistics estimates that 14.04% of the children are handicapped by deafness. Everything accomplished by the medical profession which tends to stamp out infectious diseases and all advice given by them to parents as to the care of the ears of their children, also the teaching of eugenics, will help to reduce this percentage.

EVERYBODY IS A DOCTOR

Nowadays there's little meaning
For a person to be gleaning
  When a man attaches "doctor" to his name—
He may be a chiropractor
Or painless tooth extractor—
  He's entitled to the title just the same.

Or perhaps he is a preacher
Or a lecturer or teacher,
  Or an expert who cures chickens of the pip;
He may keep a home for rummies,
Or massage fat people's tummies,
  Or specialize in ailments of the hip.

Everybody is a "doctor,"
From the backwoods herb concocter
  To the man who takes the bunions from your toes,
From the frowning dietitian
To the snappy electrician
  Who shocks you loose from all the body's woes.

So there's very little meaning
For a sufferer to be gleaning
  When a man attaches "doctor" to his name.
He may pound you, he may starve you,
He may cut your hair or carve you,
  You have got to call him Doctor just the same!

—Stoddart King, Salt Lake Tribune.

Some Pathological Conditions Causing Atrophy of Muscles In the Upper Extremity.

G. M. FLETCHER, 2T8.

THE purpose of this article is to present the salient features of a number of conditions which may produce somewhat similar manifestations in the upper limb. Three of these conditions are syringomyelia, progressive muscular atrophy of spinal origin and cervical rib.

All three of these conditions have some features which suggest involvements of the nerve structures concerned in the transmission of impulses to and from the upper limb. The fibres of the motor nerves innervating the muscles of the upper extremity arise for the most part from cells situated in the cervical segments of the spinal cord. Fibres which convey sensory impulses from the arm enter the cord in the cervical region. The importance of these two statements is that syringomyelia affects most frequently the cervical and upper dorsal segments of the cord—that the early signs of progressive muscular atrophy of spinal origin are very often found in areas supplied by nerve fibres originating from these segments—and that a cervical rib is most likely to interfere with peripheral nerves carrying fibres from the same segments.

The first of the three conditions mentioned, namely, syringomyelia, usually develops before the thirteenth year. The lesion in syringomyelia is essentially a gliosis about the central canal. Parts of the resulting mass of glial tissue undergo degeneration with subsequent cavity formation in the substance of the cord. These cavities are usually filled with fluid and vary greatly in shape and extent. They are most frequently situated in the central regions of the cord—that is near the central canal, but may involve the anterior and posterior cornua and invade the posterior columns. The cavities are more likely to be dorsal to the central canal than ventral. In addition to the mass of glial tissue and cavity formation there may be degenerative processes in the neighbouring parts of the cord due to pressure; the posterior columns particularly are likely to be damaged in this way. The gliomatous tissue which forms the basis of the lesion is thought to originate from the ependymal lining of the central canal. Small haemorrhages and foci of myelitis may be present in the lesion.

What are the clinical manifestations resulting from such a lesson? These obviously depend on what segments of the cord are involved and on what particular cells and fibres of those segments are affected. One of the commonest clinical evidences of syringomyelia is "syringomyelic dissociation of sensation." This is a condition in which the sensations of touch and pressure in the regions concerned are normal, but the sensations of pain and temperature are lost. As a result of this dissociation a patient's finger may be severely burned by his cigarette and he be quite unaware of the mishap. In attempting to explain this phenomenon it will be necessary to consider briefly the pathways by which the impulses of touch and pressure, pain and temperature pass from the periphery of the body to the cerebral cortex.

When stimuli of a nature which produce sensations of *touch and pressure* are applied to the areas supplied by say the eighth cervical and first thoracic nerves the impulses travel to

the cortex by a chain of at least three neurones. The cell body of the first neurone is situated in the spinal ganglion of the spinal nerve concerned with the transmission of the impulse; its axone divides dichotomously into a central and a peripheral branch. The peripheral branch runs in the spinal nerve to the skin and subcutaneous tissues, while the central branch enters the spinal cord by way of the dorsal root. The touch fibres are myelinated and enter the cuneate fasciculus through the medial division of the dorsal root. In the cuneate fasciculus the fibre divides into ascending and descending branches; the ascending run for varying distances in the posterior funiculus and give off numerous collaterals before they terminate in the grey matter of the spinal cord. Some of these ascending fibres reach the gracile and cuneate nuclei in the medulla oblongata. Those which terminate before reaching these nuclei do so by forming synapses with neurones of the second order in the grey substance of the posterior columns.

The cell body of the second neurone is in the posterior column of grey substance some distance above the point where the first neurone entered the cord. Its axone crosses the median plane and enters the ventral spinothalamic tract in the opposite anterior funiculus. The fibres ascend in this tract to the thalamus. There are other neurones of the second order having their cell bodies in the gracile and cuneate nuclei; they communicate with those neurones of the first order which reached their nuclei without crossing the mid-line. All the neurones of the second order end in the ventral part of the lateral thalamic nucleus. From the above it is apparent that there are two pathways in the spinal cord by which tactile impulses are conveyed to the thalamus — one is the uncrossed path in the dorsal funiculus—the other, the crossed path in the opposite anterior funiculus.

The third neurone in the touch and pressure chain has its cell body in the ventral part of the lateral nucleus of the thalamus. The axones of these cells pass by way of the thalamic radiation, through the posterior limb of the internal capsule and the corona radiata to the somesthetic area of the cerebral cortex in the posterior central gyrus. It may be noted at this point that the pathway for conduction of tactile impulses runs in the dorsal funiculus until a point is reached some segments above the level at which the fibres entered the cord.

Impulses of *pain and temperature* travel by closely related but not identical paths in the spinal cord; for convenience they are here considered together. When stimuli which produce sensations of pain and temperature are applied to the skin of the area supplied, let us say again, by the eighth cervical and first thoracic nerves, the impulses are conveyed to the cortex as was the case with impulses of touch and pressure by a chain of at least three neurones. The cell body of the first neurone is in a spinal ganglion. The central processes of the axones, etc., entral processes of the axones concerned with transmission of thermic and painful impulses are unmyelinated. They enter the spinal cord by way of the lateral division of the dorsal root and end almost at once in the substantia gelatinosa Rolandi. The cell bodies of neurones of the second order are situated in the posterior columns of grey substance at the level at which the dorsal roots enter the cord. Axones from these cells cross the mid-line and ascend in the lateral spinothalamic tract to the lateral nucleus of the thalamus. The cell bodies of the neurones of the third order are in the lateral nucleus of the thalamus and their axones ascend by way of the thalamic radiation and posterior limb of the internal capsule to the somesthetic area of the cerebral cortex. Painful impulses are perceived once they get as far as the thalamus—they do not have to reach the cortex to enter consciousness.

The explanation of the phenomenon of syringomyelic dissociation lies in the anatomical facts just reviewed. In discussing the lesion of syringomyelia it was noted that the cells of the posterior column of grey substance were frequently affected. Now, in the posterior columns of grey substance at the level at which the sensory fibres of the lower cervical and upper thoracic nerves enter the cord are the cells which are units in the chain over which impulses of pain and temperature pass. If, then, there is a syringomyelic lesion in the lower cervical segments of the cord these cells are likely to be involved and so impulses of pain and temperature entering the cord at that level are arrested. It was noted also that the fibres conveying impulses of touch and pressure do not enter the posterior columns of grey substance at the level at which they enter the cord, but proceed, instead, several segments up the cord before doing so. That is, impulses of touch and pressure entering the cord in the lower cervical segments travel through those segments in parts of the cord rarely affected by syringomyelic lesions and so are uninterrupted. So it is that the phenomenon of syringomyelic dissociation occurs.

Patients suffering from syringomyelia are apt to complain of pains of the nerve root type, particularly in the arms. Again, the lesion may attack the anterior horns of grey substance and affect those cells which are the cell bodies of the lower motor neurones to many of the muscles of the arm. As a result of the destruction of these cells certain changes occur in the muscles. They lose tone and become atrophied. The tendon reflexes are abolished and the reaction of degeneration may be elicited. When a normal muscle is stimulated directly by a suitable galvanic current a sharp, quick contraction results. If the muscle is degenerated as a result of destruction of its associated lower motor neurone, then a similar stimulus produces not a quick contraction, but one which is slow and lazy. This is the reaction of degeneration. In syringomyelia, then, paralysis and atrophy of some of the muscles of the arm is common. The muscles so affected may be such that a typical claw-hand as is found in ulnar paralysis results. Among other symptoms may be mentioned destructive whitlows with atrophy of the terminal phalanges of the fingers. Disturbances of the cervical sympathetic are common.

Cervical rib is commoner than is ordinarily supposed. It is a condition which must always be considered when a patient complains of long standing discomfort and pain in the arm. Particularly is this so if there are trophic and vascular disturbances associated with the pain. The extra rib is more often on the left side; sometimes the condition is bilateral. The size of the extra rib varies greatly. It may be but a short projecting stump or there may be a complete rib formed. Often the extra rib joins the first rib of the natural complement. In many cases where the rib is incomplete a fibrous band runs from the end of the bony projection to the first rib.

A considerable variety of signs and symptoms result from the presence of a cervical rib. If the affection be unilateral then the supra-clavicular fossa on the affected side may be noticeably fuller than the other. Often associated with this fullness there is visible pulsation because the subclavian artery is lifted upward and forwards by the rib. When the supra-clavicular fossa is palpated the rib may be felt and in many cases its shape can be clearly defined. Sometimes there is an aneurismal dilatation of the subclavian artery distal to the constriction caused by the pressure of the cervical rib. This may produce a weak pulse in the limb supplied by the artery and cause ischaemia, tingling, numbness and intermittent claudication there.

Usually another prominent manifestation of the presence of a cervical rib is atrophy of certain muscles of the forearm and hand. The atrophy becomes

apparent principally in those muscles supplied by the ulnar nerve. Among these are all the interossei, the short muscles of the ball of the thumb, the third and fourth lumbricales and the ulnar half of the flexor digitorum profundus. The wasting is apt to involve the muscles of the ball of the thumb first, so that often flattening of the thenar eminence is one of the first indications of the presence of cervical rib. As a result of the wasting of the interossei and lumbricales the spaces between the metacarpal bones become pronounced. In some cases the damage done to the eighth cervical and first thoracic nerves is of such severity that the paralysis and atrophy resulting are sufficient to produce the typical claw-hand of ulnar paralysis. Very similar changes were referred to in discussing syringomyelia.

In addition to the above, due to pressure on the fibres of the ulnar nerve there may be certain sensory phenomena. These may take the form of a continuous dull ache noticed most down the ulnar surface of the fore-arm or be only a slight feeling of numbness or a tingling sensation. Anaesthesia of certain areas of the ulnar sensory distribution are fairly common. Again, there may be a dissociation of cutaneous sensation—tactile and thermic senses being lost and that of pain retained.

Among those conditions which may cause atrophy of the muscles of the upper extremity is a group of three which affect the paths by which motor impulses travel in the spinal cord. These three conditions which are frequently described apart from one another really belong together. They are progressive muscular atrophy of spinal origin, amyotrophic lateral sclerosis and progressive bulbar paralysis. The anatomical basis of these three conditions is a slowly advancing atrophic change in the upper and lower motor neurones.

The cell bodies of the upper motor neurones are the giant pyramidal cells of the cerebral motor cortex. These cells give rise to axones which traverse the rostral half of the posterior limb of the internal capsule, the intermediate three-fifths of the cerebral peduncle, the basilar portion of the pons and the pyramid of the medulla oblongata. After undergoing a partial decussation they are continued into the spinal cord. Most of the fibres cross in the pyramidal decussation in the lower part of the medulla oblongata and continues in the opposite lateral funiculus as components of the lateral corticospinal tract. The fibres which do not cross in the medulla continue downwards in the ventral corticospinal tract; they cross the mid-line just before they terminate at various levels of the cord. All axones originating from cells in the left motor cortex end in the right anterior column of grey substance. The cell bodies of the lower motor neurones are the large multipolar cells of the anterior columns of grey substance. These cells give rise to axones that pass from the spinal cord in the ventral roots and are distributed through the spinal nerves to the skeletal musculature.

The manifestations present in each individual case depend on which neurones in the motor chain are involved first, which are involved most and whether or not both are involved simultaneously. Considering the matter from the standpoint of the nature of the paralysis present—whether spastic or flaccid—Charcot divided the cases of progressive muscular atrophy of spinal origin into two groups. In one group there was a simple atrophy of muscles and as an anatomical basis for this he believed that the ventral horns—or in other words, that the lesion producing the condition was a destructive one confined to the lower motor neurones. Such a condition he called progressive muscular atrophy of the Aran-Duchenne type. In the other group he placed cases in which there was a spastic paralysis accompanied by wasting; to this type he gave the name amyotrophic lateral sclerosis and as the anatomical basis for the condition he sug-

gested primary sclerosis of the pyramidal tract. That is, the lesion was thought to be an upper motor neurone affair with a secondary degeneration of the lower motor neurones. At present no such sharp distinction is made; it is believed in either case—whether the paralysis be flaccid or spastic—both neurones of the motor pathway are involved.

Regarding the morbid anatomy of these conditions it may be said that the essential of the condition is a slowly progressing degeneration of the motor path involving particularly the lower motor neurones, but affecting also the upper neurones either at the same time as the lower, or earlier or later in the course of the disease. In other words there is a primary progressive degeneration involving both corticospinal and spinoperipheral neurones. Examination of a cross-section of the spinal cord from a case suffering from one or other of the conditions being considered shows that there is a degeneration of the cells of the anterior cornua with atrophy of the horn itself and a degeneration of both the direct and crossed pyramidal tracts. Usually the lesion in the horns is most pronounced in the cervical region.

Accompanying the initial degeneration there is a replacement gliosis so that the picture is one of sclerosis of the pyramidal tracts and the anterior horns. The sclerosis may not be confined to the motor tracts, but may extend to the adjacent ventral and lateral tracts. In the grey substance of the medulla changes analagous to those in the anterior horns are found. The nuclei of the hypoglossal, spinal accessory, vagus, glossopharyngeal and faciae nerves are involved. This results in a glosso-labio-laryngeal paralysis or progressive bulbar paralysis. The lesion may be confined to these nuclei and no spinal symptoms be apparent. Besides these changes the fibres of the ventral nerve roots passing through the white substance are wasted. Outside the cord the ventral roots are atrophied and the cells of the motor cortex may be degenerated. The muscles supplied by the motor neurones involved in the lesion are atrophied.

The signs and symptoms presented by patients suffering from one or other of these spinal conditions vary considerably. When the lesion is such that an atonic form results—that is one in which the paralysis is flaccid, the so-called Aran-Duchenne type—the picture may be somewhat as follows: The wasting is very gradual and may be preceded by irregular pains or stiffness in the muscles involved. Usually the hands are affected early in the course of the disease and there is difficulty in performing delicate manipulations. The muscles of the ball of the thumb are among the first to show changes. Very soon the lumbricales and interossei waste and the spaces between the metacarpal bones become pronounced. As the disease progresses Duchenne's "main en griffe" results because of the contraction of the flexor and extensor muscles and the atrophy of the interossei, lumbricals and muscles of the ball of the thumb. Later the flexors of the fore-arm and then the corresponding extensors are affected. Of the muscles of the shoulder girdle the deltoid is the first to show change. The trunk muscles are gradually involved and the head falls forward because of weakness of the muscles supporting it. The muscles of the legs often remain unaffected until the atrophic changes are well marked in the arms and trunk. Late in the disease the abdominal and intercostal muscles may be involved so that the patient grows very thin of body and may be exhibited as an "attraction" at "side-shows" as a "living skeleton." Irritability of the muscles is increased and in some cases there are fibrillary contractions which may affect both the involved and the uninvolved muscles. As the disease progresses the irritability of the muscles to both galvanic and faradic stimuli gradually lessens. In some cases of rapid wasting

the reaction of degeneration may be present in the muscles. The patient may complain of coldness and numbness of the affected limbs, but sensation is unimpaired.

In a case such as Charcot designated as one of amytrophic lateral sclerosis the picture is different. There is a spastic paralysis preceding the wasting and the reflexes are greatly increased. The loss of power may be out of all proportion to the wasting, which is never so great as in the flaccid form. As has been netioned already, the symptoms and signs present in any patient suffering from these conditions depends on the relative time and relative degree of involvment of upper and lower motor neurones. The most usual finding is a flaccid, atrophic paralysis with increased reflexes.

When the sclerosis affects the motor nuclei of the medulla early or first, the condition is called bulbar paralysis. Such a paralysis, due to sclerosis, has practically no independent existence, as the cord is sooner or later involved. The signs of involvement of these nuclei are: At first there is difficulty in pronouncing dentals and linguals and so there is a slight speech defect. The paralysis starts first in the tongue and later spreads to the lips, pharyngeal muscles and vocal cords. As a result of the involvement of these structures there is difficulty in mastication and swallowing of food, drooling of saliva and feebleness of voice.

In conclusion,—this article was prepared not with the idea of considering the cause of wasting of muscles nor with the intention of presenting a mass of detail dealing with the aetiology and p a t h o l o g y of the conditions herein mentioned, but with the idea of presenting a readily assimilable arrangement of some of the more outstanding points about a few conditions which do cause wasting of muscles and which may have certain other features in common. In dealing with a number of conditions having common features a certain amount of repetition has been unavoidable.

On the Technique of Intravenous Injections.

In view of the increasing number of medicaments that are to-day administered by the intravenous route, it behoves every student of medicine to familiarize himself with the proper technique for this procedure. While the operation seems exceedingly simple it is fraught with many pitfalls, as the neophyte will soon discover.

The needle used should be as small as practicable, in order to lessen the pain of the procedure, and to leave as little of a tell-tale scar as possible; 20-27 gauge needles are suitable. Steel needles invariably rust with use, and will give rise to clotting within the lumen, and hence should be discarded before they are too old. Needles should be rinsed with alcohol after use in order to delay rusting. Platinum needles are a great convenience, can be sterilized in a flame, and never rust.

The skin should be prepared by smearing with 70% alcohol, or with decolorized tincture of iodine. Ordinary iodine obscures the blue color of the veins.

With adults the ante-cubital veins are usually the most accessible. In order to render the veins turgid, and to make them stand out, it is not always necessary to resort to the use of a tourniquet. Simply have an assistant, or the patient himself compress the upper arm with the hand, while just before inserting the needle rub the forearm vigorously, in direction from

the hand to the elbow. Direct the patient to close his fist tightly.

Glass syringes only should be used. When irritating substances such as calcium chloride or neosalvarsan are to be injected it is often useful to aspirate a little sterile water into the syringe after filling with the medicament, so that any fluid escaping from the needle into the tissues will cause no pain. The skin just below the site of injection should be drawn tight by the thumb of the left hand, and the syringe held in the right hand. The skin should then be penetrated. The veins whose position is ascertained by inspection or palpation is then approached with the needle at an angle of 20 degrees to the long axis of the vein. If the vein be approached at too acute an angle the wall is apt to be dissected, and if at too obtuse an angle the vein is apt to collapse and recede from the point of the needle. When the needle is in the vein a distinct lessening of resistance is felt by the technician, and a spurt of blood will be seen in the barrel of the syringe (hence the necessity for glass). The needle should then be brought parallel to the vein, and advanced cautiously about 5 millimeters into the vein along its lumen.

One usually fails to hit the vein because one goes too deeply rather than too superficially. In cases of failure to enter the vein, the same vein should be tried again either above or below the original site, before attempting to enter another vein.

When one is sure the vein has been entered, the pressure on the upper arm should be released, and the injection begun. This should be done slowly in all cases. The patients should be warned at this point of the probable effects of the injection (e.g. a sense of warmth and flushing in the case of calcium chloride).

It is not usually necessary to warm the solution to be injected unless it be a large injection (over 10 cc.). The heat of a freshly sterilized syringe is sufficient to raise the contained solution to body temperature, and if the solution is given slowly the temperature is of little significance.

Any sudden pain in the region of the point of the needle during the injection is an indication for stopping the injection and determining whether the needle is still in the vein. The needle should be re-inserted before the injection is proceeded with. The patient should be warned to indicate the moment he feels any stinging at the point of the needle. With many of the solutions injected intravenously the escape of any considerable amount into the tissues results in necrosis, sloughing and ulceration.

The injection can be rendered completely painless if, before withdrawal of the needle a little blood is aspirated into the syringe. This prevents the escape of any of the irritating fluid which would otherwise remain in the lumen of the needle, and which might escape into the tissues during withdrawal.

Before the needle is withdrawn a pledget of cotton should be pressed down over the wound, and the needle quickly taken out. This prevents oozing. The wound should be briskly rubbed once or twice with the cotton. Dressings are superfluous.

—M. B. C.

Translation From the Sanskrit Dealing With the Creation of Women.

In the beginning when Twastri came to the creation of women, he found he had exhausted his materials in the creation of man, so that no solid elements were left. In this dilemma, after profound meditation he did as follows. He took the rotundity of the moon, the curves of the creepers, the clinging of tendrils, the trembling of grass, the slendernes sof the reed, the bloom of the flowers, the lightness of the leaves, the glances of deer, the joyous gaiety of sunbeams, the weeping of clouds, the fickleness of the winds, the timidity of the hare, the vanity of the peacock, the sweetness of honey and the cruelty of the tiger, the warm glow of fire and the coldness of snow, the chattering of the jays, and the cooing of the doves, then compounding all these together he made woman and gave her to man. But after one week man came to him and said, "Lord, the creature you have given me makes life miserable. She chatters incessantly and teases me beyond endurance, never leaving me alone. She requires instant attention and takes up all my time. She cries about nothing and is always idle, so I have come to give her back, as I cannot live with her." So Twastri said, "Very well," and took her back. Then after another week, man came to him again and said, "Lord, I find that my life is very lonely since I gave you back that creature. I remember how she used to dance and sing to me, look at me out of the corner of her eye, and play with me and cling to me. Her laughter was music and she was beautiful to look at. Give her back to me again." So Twastri said, "Very well," and gave her back again. After three days man came and said, "Lord, I know not how it is, but after all, I have come to the conclusion she is more of a trouble than a pleasure, so please take her back again." But Twastri said, "Be off with you, I will have no more of this, you must manage how you can." The man said, "I cannot live with her," but Twastri replied, "Neither could you live without her, take her back," and the man said, "What is to be done? I cannot live either with her or without her."—(From address of Sir John Broadbent, delivered during Centenary celebration.

"Most men when they are thinking, are merely rearranging their prejudices."—Knut Rockne.

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We will follow with a great deal of interest the announcement in a recent issue of the Canadian Medical Association Journal that negotiations are under way between the Royal College of Surgeons of England and the Canadian Medical Association, with a view to holding the Primary Examinations for Fellowship in the Royal College in Canada as well as in England. If the arrangement is successfully completed both graduates and undergraduates will have the opportunity of passing the First Part of the Fellowship without going overseas. The final exam will still be held only in London.

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Try This on Your G.P.I.!

A Puzzle.—When one doctor doctors another doctor, does the doctor doing the doctoring doctor the other doctor like the doctor wants to be doctored, or does the doctor doing the doctoring doctor the other doctor like the doctor doing the doctoring wants to doctor the doctor?—The Medical Quip.

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Testimonials.

Teacher (to class in history): Now, children, what great women's letters show the suffering and hardships of her times

Chorus: Lydia Pinkham's.

—Punch Bowl.